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NEWS	5	FEB 05	German (DE) application and patent publication number format changes
NEWS	6	MAR 03	MEDLINE and LMEDLINE reloaded
NEWS	7	MAR 03	MEDLINE file segment of TOXCENTER reloaded
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L1 654916 MONOCLONAL ANTIBOD?

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L2 1 L1 AND "PDGFD"

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L2 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2004 ACS on STN
2003:551635 Document No. 139:99858 Antibodies directed to spinal cord-derived growth factor-B. Corvalan, Jose R. F.; Jia, Xiao-Chi; Feng, Xiao; Yang, Xiao-Dong; Chen, Francine; Gazit, Gadi; Weber, Richard; Bezabeh, Binyam (Abgenix, Inc., USA). PCT Int. Appl. WO 2003057857 A2 20030717, 256 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US398 20030106. PRIORITY: US 2002-41860 20020107.
AB The authors disclose fully human **monoclonal antibodies** directed to spinal cord-derived growth factor-B (SCDGFB/**PDGFD**). In one example, the antibodies are shown to neutralize the proliferative response of fibroblasts to a p53 fragment of SCDGFB/**PDGFD**. In a second example, the antibodies comprise reagents for the detection of SCDGFB/**PDGFD** in blood in various malignancies.

=> s l1 and platelet derived growth factor D
L3 10 L1 AND PLATELET DERIVED GROWTH FACTOR D

=> dup remove l3
PROCESSING COMPLETED FOR L3
L4 4 DUP REMOVE L3 (6 DUPLICATES REMOVED)

=> d l4 1-4 cbib abs

L4 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2004 ACS on STN
2003:551635 Document No. 139:99858 Antibodies directed to spinal cord-derived growth factor-B. Corvalan, Jose R. F.; Jia, Xiao-Chi; Feng, Xiao; Yang, Xiao-Dong; Chen, Francine; Gazit, Gadi; Weber, Richard; Bezabeh, Binyam (Abgenix, Inc., USA). PCT Int. Appl. WO 2003057857 A2 20030717, 256 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM,

AZ, BY; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US398 20030106. PRIORITY: US 2002-41860 20020107.

AB The authors disclose fully human **monoclonal antibodies** directed to spinal cord-derived growth factor-B (SCDGFB/PDGFD). In one example, the antibodies are shown to neutralize the proliferative response of fibroblasts to a p53 fragment of SCDGFB/PDGFD. In a second example, the antibodies comprise reagents for the detection of SCDGFB/PDGFD in blood in various malignancies.

L4 ANSWER 2 OF 4 MEDLINE on STN DUPLICATE 1

2003398231. PubMed ID: 12937299. A fully human **monoclonal antibody** (CR002) identifies PDGF-D as a novel mediator of mesangioproliferative glomerulonephritis. Ostendorf Tammo; van Roeyen Claudia R C; Peterson Jeffrey D; Kunter Uta; Eitner Frank; Hamad Avin J; Chan Gerlinde; Jia Xiao-Chi; Macaluso Jennifer; Gazit-Bornstein Gadi; Keyt Bruce A; Lichenstein Henri S; LaRochelle William J; Floege Jurgen. (Division Nephrology, University of Aachen, Germany.) Journal of the American Society of Nephrology : JASN, (2003 Sep) 14 (9) 2237-47. Journal code: 9013836. ISSN: 1046-6673. Pub. country: United States. Language: English.

AB PDGF-B is of central importance in mesangioproliferative diseases. PDGF-D, a new PDGF isoform, like PDGF-B, signals through the PDGF betabeta-receptor. The present study first determined that PDGF-D is mitogenic for rat mesangial cells and is not inhibited by a PDGF-B antagonist. Low levels of PDGF-D mRNA were detected in normal rat glomeruli. After induction of mesangioproliferative nephritis in rats by anti-Thy 1.1 mAb, glomerular PDGF-D mRNA and protein expression increased significantly from days 4 to 9 in comparison with nonnephritic rats. Peak expression of PDGF-D mRNA occurred 2 d later than peak PDGF-B mRNA expression. In addition, PDGF-D serum levels increased significantly in the nephritic animals on day 7. For investigating the functional role of PDGF-D, neutralizing fully human mAb were generated using the XenoMouse technology. Rats with anti-Thy 1.1-induced nephritis were treated on days 3 and 5 with different amounts of a fully human PDGF-DD-specific neutralizing mAb (CR002), equal amounts of irrelevant control mAb, or PBS by intraperitoneal injection. Specific antagonism of PDGF-D led to a dose-dependent (up to 67%) reduction of glomerular cell proliferation. As judged by double immunostaining for 5-bromo-2'-deoxyuridine and alpha-smooth muscle actin, glomerular mesangial cell proliferation was reduced by up to 57%. Reduction of glomerular cell proliferation in the rats that received CR002 was not associated with reduced glomerular expression of PDGF-B mRNA. PDGF-D antagonism also led to reduced glomerular infiltration of monocytes/macrophages (day 5) and reduced accumulation of fibronectin (day 8). In contrast, no effect was noted in normal rats that received an injection of CR002. These data show that PDGF-D is overexpressed in mesangioproliferative states and can act as an auto-, para-, or even endocrine glomerular cell mitogen, indicating that antagonism of PDGF-D may represent a novel therapeutic approach to mesangioproliferative glomerulonephritides.

L4 ANSWER 3 OF 4 MEDLINE on STN DUPLICATE 2

2002242895. PubMed ID: 11980634. **Platelet-derived growth factor D**: tumorigenicity in mice and dysregulated expression in human cancer. LaRochelle William J; Jeffers Michael; Corvalan Jose R F; Jia Xiao-Chi; Feng Xiao; Vanegas Sandra; Vickroy Justin D; Yang Xiao-Dong; Chen Francine; Gazit Gadi; Mayotte Jane; Macaluso Jennifer; Rittman Beth; Wu Frank; Dhanabal Mohan; Herrmann John; Lichenstein Henri S. (CuraGen Corp., Branford, Connecticut 06405, USA.. wlarochelle@curagen.com) . Cancer research, (2002 May 1) 62 (9) 2468-73. Journal code: 2984705R. ISSN: 0008-5472. Pub. country: United States. Language: English.

AB Platelet-derived growth factor (PDGF) has been directly implicated in developmental and physiological processes, as well as in human cancer and other proliferative disorders. We have recently isolated and characterized a novel protease-activated member of the PDGF family, PDGF D. PDGF D has been shown to be proliferative for cells of mesenchymal origin, signaling through PDGF receptors. Comprehensive and systematic PDGF D transcript analysis revealed expression in many cell lines derived from ovarian, renal, and lung cancers, as well as from astrocytomas and medulloblastomas. beta PDGF receptor profiling further suggested autocrine signaling in several brain tumor cell lines. PDGF D transforming ability and tumor formation in SCID mice was further demonstrated. Exploiting a sensitive PDGF D sandwich ELISA using fully human **monoclonal antibodies**, PDGF D was detected at elevated levels in the sera of ovarian, renal, lung, and brain cancer patients. Immunohistochemical analysis confirmed PDGF D localization to ovarian and lung tumor tissues. Together, these data demonstrate that PDGF D plays a role in certain human cancers.

L4 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2004 ACS on STN
2001:300544 Document No. 134:325212 Method of treating fibrosis. Gilbertson, Debra G. (Zymogenetics, Inc., USA). PCT Int. Appl. WO 2001028586 A1 20010426, 70 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US29270 20001023. PRIORITY: US 1999-PV161653 19991021; US 1999-PV165255 19991112; US 2000-PV222223 20000801.
AB Materials and methods for treating fibrosis in a mammal are disclosed. The methods comprise administering to a mammal a composition comprising a therapeutically effective amount of a zvegf3 antagonist in combination with a pharmaceutically acceptable delivery vehicle. Zvegf3 antagonists include anti-zvegf3 antibodies, mitogenically inactive receptor-binding zvegf3 variant polypeptides, and inhibitory polynucleotides. Within one embodiment of the invention the fibrosis is liver fibrosis.

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gazit g?/au or weber r?/au or bezabeh b?/au)
L5       54977 (CORVALAN J?/AU OR JIA X?/AU OR FENG X?/AU OR YANG X?/AU OR
               CHEN F?/AU OR GAZIT G?/AU OR WEBER R?/AU OR BEZABEH B?/AU)

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=> s l6 and platelet derived growth factor
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=> dup remove l7
PROCESSING COMPLETED FOR L7
L8           7 DUP REMOVE L7 (11 DUPLICATES REMOVED)

=> d l8 1-7 cbib abs
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L8 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2004 ACS on STN
2003:551635 Document No. 139:99858 **Antibodies** directed to spinal cord-derived growth factor-B. **Corvalan, Jose R. F.; Jia, Xiao-Chi; Feng, Xiao; Yang, Xiao-Dong; Chen, Francine; Gazit, Gadi; Weber, Richard; Bezabeh, Binyam** (Abgenix, Inc., USA). PCT Int. Appl. WO

2003057857 A2 20030717, 256 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US398 20030106. PRIORITY: US 2002-41860 20020107.

AB The authors disclose fully human monoclonal **antibodies** directed to spinal cord-derived growth factor-B (SCDGFB/PDGFD). In one example, the **antibodies** are shown to neutralize the proliferative response of fibroblasts to a p53 fragment of SCDGFB/PDGFD. In a second example, the **antibodies** comprise reagents for the detection of SCDGFB/PDGFD in blood in various malignancies.

L8 ANSWER 2 OF 7 MEDLINE on STN DUPLICATE 1

2003398231. PubMed ID: 12937299. A fully human monoclonal **antibody** (CR002) identifies PDGF-D as a novel mediator of mesangioproliferative glomerulonephritis. Ostendorf Tammo; van Roeyen Claudia R C; Peterson Jeffrey D; Kunter Uta; Eitner Frank; Hamad Avin J; Chan Gerlinde; **Jia Xiao-Chi**; Macaluso Jennifer; Gazit-Bornstein Gadi; Keyt Bruce A; Lichenstein Henri S; LaRochelle William J; Floege Jurgen. (Division Nephrology, University of Aachen, Germany.) Journal of the American Society of Nephrology : JASN, (2003 Sep) 14 (9) 2237-47. Journal code: 9013836. ISSN: 1046-6673. Pub. country: United States. Language: English.

AB PDGF-B is of central importance in mesangioproliferative diseases. PDGF-D, a new PDGF isoform, like PDGF-B, signals through the PDGF betabeta-receptor. The present study first determined that PDGF-D is mitogenic for rat mesangial cells and is not inhibited by a PDGF-B antagonist. Low levels of PDGF-D mRNA were detected in normal rat glomeruli. After induction of mesangioproliferative nephritis in rats by anti-Thy 1.1 mAb, glomerular PDGF-D mRNA and protein expression increased significantly from days 4 to 9 in comparison with nonnephritic rats. Peak expression of PDGF-D mRNA occurred 2 d later than peak PDGF-B mRNA expression. In addition, PDGF-D serum levels increased significantly in the nephritic animals on day 7. For investigating the functional role of PDGF-D, neutralizing fully human mAb were generated using the XenoMouse technology. Rats with anti-Thy 1.1-induced nephritis were treated on days 3 and 5 with different amounts of a fully human PDGF-DD-specific neutralizing mAb (CR002), equal amounts of irrelevant control mAb, or PBS by intraperitoneal injection. Specific antagonism of PDGF-D led to a dose-dependent (up to 67%) reduction of glomerular cell proliferation. As judged by double immunostaining for 5-bromo-2'-deoxyuridine and alpha-smooth muscle actin, glomerular mesangial cell proliferation was reduced by up to 57%. Reduction of glomerular cell proliferation in the rats that received CR002 was not associated with reduced glomerular expression of PDGF-B mRNA. PDGF-D antagonism also led to reduced glomerular infiltration of monocytes/macrophages (day 5) and reduced accumulation of fibronectin (day 8). In contrast, no effect was noted in normal rats that received an injection of CR002. These data show that PDGF-D is overexpressed in mesangioproliferative states and can act as an auto-, para-, or even endocrine glomerular cell mitogen, indicating that antagonism of PDGF-D may represent a novel therapeutic approach to mesangioproliferative glomerulonephritides.

L8 ANSWER 3 OF 7 MEDLINE on STN DUPLICATE 2

2002242895. PubMed ID: 11980634. **Platelet-derived growth factor** D: tumorigenicity in mice and dysregulated expression in human cancer. LaRochelle William J; Jeffers Michael; **Corvalan Jose R F; Jia Xiao-Chi; Feng Xiao;** Vanegas Sandra; Vickroy Justin D; **Yang Xiao-Dong; Chen**

Francine; Gazit Gadi; Mayotte Jane; Macaluso Jennifer; Rittman Beth; Wu Frank; Dhanabal Mohan; Herrmann John; Lichenstein Henri S. (CuraGen Corp., Branford, Connecticut 06405, USA.. wlarochelle@curagen.com) . Cancer research, (2002 May 1) 62 (9) 2468-73. Journal code: 2984705R. ISSN: 0008-5472. Pub. country: United States. Language: English.

AB **Platelet-derived growth factor** (PDGF) has been directly implicated in developmental and physiological processes, as well as in human cancer and other proliferative disorders. We have recently isolated and characterized a novel protease-activated member of the PDGF family, PDGF D. PDGF D has been shown to be proliferative for cells of mesenchymal origin, signaling through PDGF receptors. Comprehensive and systematic PDGF D transcript analysis revealed expression in many cell lines derived from ovarian, renal, and lung cancers, as well as from astrocytomas and medulloblastomas. beta PDGF receptor profiling further suggested autocrine signaling in several brain tumor cell lines. PDGF D transforming ability and tumor formation in SCID mice was further demonstrated. Exploiting a sensitive PDGF D sandwich ELISA using fully human monoclonal **antibodies**, PDGF D was detected at elevated levels in the sera of ovarian, renal, lung, and brain cancer patients. Immunohistochemical analysis confirmed PDGF D localization to ovarian and lung tumor tissues. Together, these data demonstrate that PDGF D plays a role in certain human cancers.

L8 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2004 ACS on STN

2001:136991 Document No. 134:198075 Triglyceride-free compositions and methods for enhanced absorption of hydrophilic therapeutic agents. Patel, Mahesh V.; **Chen, Feng-Jing** (Lipocine, Inc., USA). PCT Int. Appl. WO 2001012155 A1 20010222, 113 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US18807 20000710. PRIORITY: US 1999-375636 19990817.

AB The present invention relates to triglyceride-free pharmaceutical compns., pharmaceutical systems, and methods for enhanced absorption of hydrophilic therapeutic agents. The compns. and systems include an absorption enhancing carrier, where the carrier is formed from a combination of at least two surfactants, at least one of which is hydrophilic. A hydrophilic therapeutic agent can be incorporated into the composition, or can be co-administered with the composition as part of a pharmaceutical system. The invention also provides methods of treatment with hydrophilic therapeutic agents using these compns. and systems. For example, when a composition containing Cremophor RH40 0.30, Arlacel 186 0.20, Na taurocholate 0.18, and propylene glycol 0.32 g, resp., was used, the relative absorption of PEG 4000 as a model macromol. drug was enhanced by 991%.

L8 ANSWER 5 OF 7 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

2002:275121 Document No.: PREV200200275121. Interaction between macrophage and smooth muscle progenitor cells in vascular remodeling lesions in mice. **Yang, Xu** [Reprint author]; Yokode, Masayuki [Reprint author]; Sano, Hideto [Reprint author]; Kataoka, Hiroshi; Murayama, Toshinori; Zhuge, Xin; Kita, Toru. Graduate Sch of Med, Kyoto Univ, Kyoto, Japan. Circulation, (October 23, 2001) Vol. 104, No. 17 Supplement, pp. II.299. print.
Meeting Info.: Scientific Sessions 2001 of the American Heart Association. Anaheim, California, USA. November 11-14, 2001. American Heart Association.

CODEN: CIRCAZ. ISSN: 0009-7322. Language: English.

L8 ANSWER 6 OF 7 MEDLINE on STN DUPLICATE 3

2000491409. PubMed ID: 11043859. Fatty acids modulate protein kinase C activation in porcine vascular smooth muscle cells independently of their effect on de novo diacylglycerol synthesis. Lu X; **Yang X Y;** Howard R L; Walsh J P. (Department of Biochemistry and Molecular Biology, Indiana University School of Medicine, Indianapolis, USA.) Diabetologia, (2000 Sep) 43 (9) 1136-44. Journal code: 0006777. ISSN: 0012-186X. Pub. country: GERMANY: Germany, Federal Republic of. Language: English.

AB AIMS/HYPOTHESIS: Diabetes-induced activation of protein kinase C has been associated with the development of vascular complications. Elevated de novo diacylglycerol synthesis has been postulated to underlie this protein kinase C activation. Diabetes also increases the circulating concentrations of non-esterified fatty acids, which are immediate precursors of diacylglycerol through the de novo pathway. We hypothesized that increased fatty acids contribute to de novo diacylglycerol synthesis and activation of protein kinase C in vascular cells. METHODS: Primary cultures of porcine carotid smooth muscle cells were exposed to fatty acids, bound to albumin in physiologic ratios. Diacylglycerol and triacylglycerol were measured in extracts of these cells. Protein kinase C activation was measured as membrane translocation with isoform-specific **antibodies**. RESULTS: Saturated fatty acids caused considerable accumulation of diacylglycerol through de novo synthesis. Unsaturated fatty acids increased triacylglycerol, but not diacylglycerol. **Platelet-derived growth factor** activated the alpha, epsilon and zeta protein kinase C isoforms. Activation of the alpha and zeta isoforms was amplified by oleate pretreatment but inhibited by palmitate. In the absence of growth factor stimulation, neither palmitate nor oleate had any effect on the membrane/cytosol distribution of any protein kinase C isoform. CONCLUSION/INTERPRETATION: Saturated fatty acids elicited de novo diacylglycerol synthesis in vascular smooth muscle cells without activating protein kinase C. Effects of fatty acids on protein kinase C activation by **platelet-derived growth factor** did not correlate with the effects on de novo diacylglycerol synthesis. These results indicate that de novo diacylglycerol synthesis is, by itself, insufficient to activate protein kinase C.

L8 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2004 ACS on STN

1997:6813 Document No. 126:43545 Expression of cytokines in labial gland of Sjogren's syndrome. Ding, Dacheng; Ong, Yi; Li, Shengde; Li, Xiaochun; **Yang, Xiaojie;** Gan, Xiaodan (Dep. Immunology, Peking Union Medical College Hospital, Beijing, 100730, Peop. Rep. China). Zhonghua Yixue Zazhi, 76(6), 427-430 (Chinese) 1996. CODEN: CHHTAT. ISSN: 0376-2491. Publisher: Zhonghua Yixue Zazhi.

AB The mRNA of the following cytokines: TNFα, IL-1β, IL-6, PDGFα and PDGFβ in 24 labial gland biopsied sections of patients with Sjogren's syndrome (SS) were detected by in situ non-isotope double hybridization method. No cytokine were detected in the normal labial glands sections. Cytokine gene expression was higher in the SS patients than that in the control. The intensity of TNFα expression was 3-4 fold more in primary SS than secondary SS. The different cytokines were expressed with the different grade of lymphocytic focus. The coexpression of mRNA of TNFα and IL-1β or TNFα and IL-6 was only seen in the inflammatory mononuclear cells but not others. The expression of TNFα in acinar cells could occur earlier than the infiltration of lymphocytes in the interstitial region. The IL-6-pos. group had higher positivity of serum ANA compared with the IL-6-neg. group according to Ridit statistical anal. The serum anti-SSA and anti-SSb **antibody** had an increase tendency in the IL-6 pos. group.

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L11           0 "GFGYSYNYDYYYGMDV"

=> s "RASQSVSSSYLA"
L12           0 "RASQSVSSSYLA"

=> s "ATSSRAT"
L13           0 "ATSSRAT"

=> s "QQYGSSPCS"
L14           0 "QQYGSSPCS"

=> s human D5-18 family gene
L15           0 HUMAN D5-18 FAMILY GENE

=> s human D5 gene
L16           0 HUMAN D5 GENE

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DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
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CA SUBSCRIBER PRICE                                    -4.16      -4.16

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